servative process in which the leucocytes are thrown out in great numbers, to destroy bacteria, and as a barrier to systemic infection. The peculiar relation of the bacilli to the nuclei in the giant cells themselves, suggests the possibility of an antagonism between the two. As the pathogenic qualities of infectious germs may in many instances be abrogated or diminished at will, as in the preliminary manipulation of bacteria used in vaccinations, by placing them under peculiar conditions of environment which rob them of their virulence, or modify it, so may the resisting power of their natural enemies, the cells of the body, be diminished or increased by the same means. Watson Cheyne,[1] in a review of some of his recent experiments, after describing the manner in which large quantities of putrefactive and non-pathogenic bacteria are destroyed when injected into the blood-current of rabbits, adds "but this does not occur if the animal is in a bad state of health."

Viewed in this light, the teachings of clinical medicine, the knowledge obtained by a study of the therapeutics of the disease, and the proof offered by experimental research, are in perfect accord. All measures which tend to increase the vitality of the body cells have been found to be precisely those which are most effectual in combating tuberculosis; one by one, specific methods of treatment, which for a season enjoyed popularity, have fallen into disuse, and hygiene, climate, and feeding—in other words, a favorable environment—have alone given results which have stood the test of time.

A consideration of the evidence offered by this study, therefore, teaches that though environment may bear but the relation of a predisposing cause to microbic infection, it is, nevertheless, a potent factor in determining the future type, and even the final results of the disease, and that if we may not under-estimate the pathogenic properties of the bacillus, the effect of extremes of environment on the resisting power of the cells of the body is an element in this complex problem which should not be ignored.

THE PUPIL IN ITS SEMEIOLOGICAL ASPECTS.

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Many observations have been made, from numerous standpoints, regarding pupillary conditions, yet with a few notable exceptions, they have been studied in an isolated manner, relative to the particular disease or lesion of which they might be more or less symptomatic. They have been often looked upon, and are still regarded by some, as curious, interesting, but erratic phenomena, far too variable to be depended upon, and without any connecting thread upon which these conditions, as seen in a variety of diseases, could be strung. Few attempts have been made to grasp pupillary manifestations as a whole, still fewer to reduce the varying phenomena to principles or to reduce the laws by which they are controlled.

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[1] Bacteriology. Amer. Journ. Med. Sci., January, 1887.

Among the papers dealing with the pupil as a whole, that of Dr. Alph. Drouin is one of the most complete, and the writer is indebted to that work for information, especially from the section devoted to anatomy and physiology. Mr. Hutchinson's excellent papers on the "Symptom Significance of Different States of the Pupil," published in the first volume of *Brain*, contain much that is of value, to which the reader's attention is directed.

In the present paper it is intended to give, first, with the view of refreshing the mind of the reader, a brief outline of the physiological phenomena pertaining to the movements of the pupil, and thereafter to present a series of personal observations from which certain conclusions have been drawn.

PHYSIOLOGICAL OUTLINE.—The optic nerves have no direct control over the pupil. The impression produced by light falling upon the retina passes through the second nerves to the corpora quadrigemina, which communicate an impulse to the oculomotor centres, and so cause pupillary contraction. The second is thus the afferent nerve in this reflex (excito-motor) action. This reflex phenomenon may be brought about not only by the impression of light upon the retina, but also by mechanical stimulation of the optic nerves, which induces in them a sensation of light. When both optic nerves are divided or paralyzed, dilatation and immobility of the pupils ensue. Paralysis of one optic nerve produces dilatation of the pupil with blindness on the same side as the lesion; but owing to the preservation of consensual movements the pupil of the injured eye contracts when impressions of light are made upon the sound retina, though these are quicker and stronger on the sound side. Therefore, pupillary phenomena are sometimes wanting when only one optic is paralyzed, owing to the preservation of consensual movements; in this way it differs from unilateral lesions of the third, which always affect the corresponding pupil in a very definite way. Section or destruction of one optic tract produces dilatation of the opposite pupil, and blindness of the opposite eye.

The third nerve takes origin beneath the floor of the aqueduct of Sylvius, and supplies the ciliary muscle and sphincter of the iris, besides all the muscles of the globe with the exception of the external rectus and superior oblique. When the third nerve is stimulated contraction of the pupil on the same side ensues, and when it is divided or paralyzed the pupil dilates; it no longer responds to the action of light, and accommodation is abolished. When the central portion of a cut oculomotor is stimulated, the opposite pupil becomes contracted, provided the nerve on the opposite side remains intact. In mydriasis as a sequent of oculomotor paralysis, the pupil may be made to dilate still further by either stimulating the sympathetic, or by the action of atropine. In man, pathological lesions of the third nerve generally result in mydriasis, though the writer has seen a case of a contracted pupil due to neuritis of the third on the same side; and M. Voisin found in one instance, in the case of a general paralytic who had unequal pupils, one of which was contracted, that there was an effusion of blood into the nucleus of origin of the third on the same side. Besides its action on the pupil, paralysis of the third when complete causes paralytic ptosis, strabismus outward and downward, slight protrusion, and immobility of the eyeball. There is, of course, inability to adduct the eyeball, and consequent double vision. It is well to recollect that paralysis may be confined to individual branches of the nerve. Unilateral affections of the third show themselves in the pupil on the same side as the lesion in the brain. Intracranial unilateral section of the fifth nerve causes on the same side as the lesion, fixity of the eyeball, contraction and immobility of the pupil. The want of sensibility soon leads to inflammation and ultimate destruction of the eyeball. The same pupillary effect is produced on section of the sympathetic, and some suppose that the action of the fifth on the pupil is due to the vasomotor filaments which it contains.

The centre for dilatation of the pupil is situated in the medulla oblongata. Thence the fibres make their exit partly through the trigeminus, partly through the lateral columns of the cord, in the ciliospinal region (from the sixth cervical to the third dorsal vertebræ), passing out by the two lowest cervical and the first two dorsal nerves into the cervical sympathetic. The latter fibres find their way to the iris, not only through the cervical sympathetic, but also by the cavernous plexus, lenticular ganglion, and ciliary nerves. It may be interesting to note, as having a bearing on the question of the power of the vasomotor system on the iris, that Landois and Stirling state that though the vasomotor and oculo-pupillary fibres "lie in the same trunk in the neck they do not issue from the cord by the same nerve roots; the latter come out of the cord with the anterior roots of the first and second dorsal nerves (dog), while section of the cord between the second and fourth dorsal vertebræ produces the vasomotor changes only."[1] It may, however, be difficult to ascertain whether the latter sections produce direct pupillary effects, as the pupil may be influenced by the state of the bloodvessels. More definite information on this point is necessary.

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[1] Ferrier states that in the monkey the second dorsal is the only nerve issuing from the ciliospinal axis which produces dilatation of the pupil on irritation.

When the cervical sympathetic is stimulated, dilatation of the pupil ensues; and when it is divided or paralyzed, the pupil contracts. Similar results follow like causes applied to the medulla, and especially to the ciliospinal zone. Budge describes this as the centre for the dilatation of the pupil, and it may be looked upon as a secondary centre. If it be admitted that there are radiating fibres in the iris, then these are innervated by the sympathetic, causing dilatation of the pupil, while the oculomotor innervates the circular fibres. But Rouget, and many others, do not admit the existence of radiating fibres, explaining the action of the cilio-spinal axis on the pupil as being due solely to its vasomotor control.

When the cervical sympathetic is divided, the vessels of the corresponding side of the head, face, ear, and mucous membrane of the mouth, become dilated; there is sweating on that side of the head, elevation of temperature, the vessels of the iris are engorged with blood, and there is contraction of the pupil. If the peripheral end of the cut sympathetic be electrically stimulated all these phenomena disappear, and the pupil dilates. Many infer from these and other phenomena, that the action of the sympathetic on the pupil is exercised through the intermediary of the bloodvessels.

Unilateral excitation of the cord in the ciliospinal region produces dilatation of the pupil on the same side. Although the ciliospinal zone is specially susceptible to impressions giving rise to pupillary phenomena, yet irritation applied to almost any part of the cord is apt to be followed by dilatation of the pupil. This centre is likewise connected with the afferent sensory nerves, so that painful impressions on the skin and mucous membranes may reflexly produce dilatation of the pupils.

When the corpora quadrigemina are irritated, both pupils contract, and when destroyed, dilatation with immobility of pupils and destruction of sight ensue. It will be observed, that the pupillary effect is similar to that produced by lesions of the third nerve.

Were Longet's observation correct, one could remove from an animal the whole brain, with the exception of the corpora quadrigemina, without producing any pupillary indication. The writer would like to have further evidence on this point before accepting this statement as a fact.[1] Even were it the case, it would, as M. Drouin points out, not be correct to conclude that lesions of the cortical masses have no effect upon the movements of the iris, as various encephalic modifications have distinctive pupillary indications. It is supposed that there are two cortical motor centres for the pupil; the one situated in the base of the first and second frontal convolutions, which, on irritation, causes dilatation of the pupil through the ciliospinal subcentre; the other located in the angular gyrus, connected with the nucleus of the third nerve by means of centrifugal fibres, which, when irritated, causes contraction of the iris with convergent eyes. These two centres in the cortex of the brain, stated to be connected so precisely, the one with the spinal centre, the other with the oculomotor beneath the aqueduct of Sylvius, are not yet definitely established, and in some measure are yet inferential.

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[1] Ferrier states that in animals deprived of their cerebral hemispheres, a flash of light before their eyes will cause the pupils to contract. Judging from this, the pupils must be either medium or dilated in such cases.

In considering the manner in which cerebral lesions may act upon the pupils, it is necessary to weigh the possible participation of the various nerve centres, and the effects of modifications of the brain as a whole upon them, and also the relation of the state of the cerebral circulation to that of the iris.

The phenomena produced by lesions of the cerebellum upon the pupil are varied, generally myosis, occasionally mydriasis; both being accompanied by strabismus.

Meningeal affections may extend so widely that many parts of the brain and basal nerves may be implicated; and probably by reflex sympathetic action, changes may be induced in the eye and iris. When one remembers that the internal carotid supplies both the pia mater and globe of the eye, and that the same vasomotor centre supplies the vascular nerves of those arterial branches, the influence of the meningeal affections over the iris may be understood.

There is considerable divergence of opinion regarding the manner in which certain pupillary movements are brought about, and also as to the exact mechanism controlling these movements. While all admit the existence of the sphincter of the iris, there is no unanimity as to the mode by which dilatation is effected. While some believe that there are muscular radiating fibres whose function it is to dilate the iris, others deny their existence, believing that the dilatation and contraction of accommodation take place in virtue of the inherent elasticity of the iris. Those holding the latter view say, physiology shows that the movements of the iris are controlled in two ways, the one being functional or active, relating to accommodation during the exercise of vision, the other being passive, depending on the variations of the circulation in the encephalon and iris. The former movements are those arising from the impressions of light on the retina communicated through the second nerve to the corpora quadrigemina, which produce contraction of the pupil through the oculomotor acting on the iris. In those functional movements the changes in the pupil are brought about by the elasticity of the iris. The passive or vascular alterations of calibre are quite of a different character, and are independent of visual impressions, though the power of accommodation may remain. In those the movements of the pupil are regulated by the vascular system of the iris, which is in complete harmony with that of the encephalon. Myosis ensues when the vessels of the encephalon and those of the iris are dilated; and dilatation of the pupil corresponds to anæmia of the encephalon and iris. It is in this way that congestion of the brain and its membranes, the result of paralysis of the cervical sympathetic, produces fluxion or paralytic dilatation of the vessels of the iris and contraction of the pupil; while in irritation of the sympathetic, in ischæmia of the brain, due to epilepsy, syncope, strong moral emotions, powerful irritation of the sensory nerves, etc., the vessels of the iris being contracted, mydriasis ensues. Regarding the action the various states of the encephalon might have on the pupils, it is said that one has only to determine what effect the lesions have on the circulation. Prominent among those who have advocated this view is Mosso, of Turin, who declares that the size of the pupil follows exactly the oscillations of the bloodvessels, and M. Drouin warmly espouses the same view, regarding the pupil as the manometer of the cerebral circulation. M. François Frank refutes this theory, and states that the movements of the pupil are quite independent of the condition of the bloodvessels; the evidence he advances in support is far from conclusive.

There is this difference between the influence exerted on the pupil by cerebral affections and those arising from the ciliospinal axis, the former can affect the size of the pupil in two ways: first, through the medium of the bloodvessels, second, by injuring the apparatus controlling the functional movements of the iris (the optic and oculomotor nerves and corpora quadrigemina); while the latter, the ciliospinal, can affect the pupils in only one way, by means of the reflex phenomena determining dilatation and contraction of the vessels.

METHOD OF EXAMINING THE PUPIL.—In examining the pupil, the shape, size, activity, and any difference existing between the two pupils, should be noted. The following rules ought to be attended to. The patient ought to face some source of light, such as a window.

1. Both eyelids ought to be closed, and after the lapse of a few seconds both ought to be simultaneously opened, when the observer should note whether they contract equally.

2. One eyelid should then be closed for half a minute; observe on opening it whether there be immediate contraction in that pupil, and whether simultaneous contraction takes place in the opposite pupil. The same should be repeated in the opposite eye. By this means an amaurotic eye would be detected, as in it the consensual movement persists, while the direct movements are abolished.

Pupilometers. Many pupilometers have been invented, such as those of Olbers, Follin, Fick, Badel, Galezowski, Landolt, Gorham, etc., some of them being ingenious instruments. For practical purposes, however, Hutchinson's idea of the disk of polished steel, with holes punched in it, or the simple narrow white card, having marked on it a series of black dots, measuring from one to nine millimetres, is sufficient. When in use it is placed close to the eye, and the dot which corresponds most to the size of the pupil is noted, and its exact size in millimetres is read off. This has been employed by M. Drouin, and Dr. Finlayson figures it in his work on clinical medicine. The widest dilatation of the iris never exceeds nine millimetres.

PERSONAL OBSERVATIONS ON THE PUPILS.—The data upon which the following remarks are founded are based upon personal experience. The various facts were gleaned and recorded as isolated phenomena. It was only in reviewing and collating these, the conception evolved, that, besides the effect on the pupil of lesions of certain brain and spinal centres and their nerves, there were general cerebral conditions, which were accompanied by definite pupillary phenomena, with sufficient constancy to enable conclusions to be formulated regarding them. When these pupillary states are fenced with proper reservations, they form valuable aids to the diagnosis of some of the general conditions of the brain.

When the function of the brain is temporarily suspended, the pupils are in a state of stabile mydriasis. It matters not what the cause of this suspension of function may be, the pupillary phenomena are identical as long as this condition lasts.

In peripheral injuries, involving large extents of surface, such as the mangling of both thighs by a railway accident, a state of shock is frequently induced, in which, along with complete insensibility and the usual accompanying phenomena, the pupils are widely dilated and fixed. Reaction in such cases is often heralded by an alteration in the pupillary state, the mydriasis giving way somewhat, the pupil at the same time becoming mobile, and soon after, vomiting or other sign of reaction sets in. The same phenomena are seen when shock is induced by blows upon the epigastrium, by severe pain, such as may be occasioned by injury of the testicle, and by powerful mental impressions. Again, when, instead of the injury being peripheral, it is applied to the brain itself, and is of sufficient severity to cause suspension of the cerebral function, the pupils are widely dilated and fixed.

As to this state of central shock or cerebral concussion, writers are at variance with one another concerning the pupillary condition attending it. Some state that the pupils are dilated, others that they are contracted, most that they are variable, while a few state that they are unequal. Hospital surgeons, who make a daily or a less frequent visit to their wards, have few opportunities of seeing uncomplicated cases of concussion of the brain, as in the majority of instances the phenomena attending this condition pass off within a period of hours, and in many before the patient reaches the hospital. As an "interne" in the Royal Infirmary, the writer witnessed a number of such cases, the symptoms in each of these passing off in a few hours; and afterward, while occupying the post of Casualty Surgeon in Glasgow, the writer had the fortune of seeing, and the opportunity of observing to a conclusion, at least twenty well-marked instances. The data thus derived pointed clearly to the conclusion that in uncomplicated cases of concussion the pupils are widely dilated and fixed. One of the earliest indications of recovery from this state is to be found in the pupils, the wide dilatation relaxing somewhat, accompanied by a slight mobility. During the period of rallying the pupil is very variable, and generally more or less contracted. In cases of concussion, which are complicated by brain lesion, such as slight compression, the pupillary phenomena, the result of concussion, remain until the latter state passes off, when the slight compression manifests itself, among other ways, by a pupillary state, such as inequality, contraction, etc. In this way, concussion may temporarily mask other and more serious, though more limited brain lesions, which may manifest themselves when it has subsided. If these two conditions are not discriminated, the pupillary phenomena of the one may be attributed to that of the other.

In hemorrhage considerable loss of blood may be borne without inducing marked pupillary changes, as long as the blood be not removed from the body so rapidly as to induce syncope. When, however, the amount of blood lost is great, equalling one-seventh of the whole, the cerebral irritability is increased, and is accompanied by myosis. When the depletion is carried much further, so that the amount lost is equal to one-fifth of the whole, the cerebral anæmia is so great as seriously to reduce the functional power of the brain; then the pupils are dilated, and if this depletion be pushed so as to induce syncope, they become widely dilated and fixed. When they are in this condition, it is interesting to note how quickly they undergo change when the cerebral anæmia is dispelled by lowering the head and raising the limbs, so as to empty the latter of their vascular contents, and thereby replenishing the heart and brain. The pupils then become mobile, less dilated, and may fall to medium size, or even become contracted, according to the amount of blood thrown into the cranium. This is quite in accordance with the observation of B. Séquard, who has shown that when an animal is suspended by the hind legs, contraction of the pupil occurs, comparable to that obtained by ablation of the superior cervical ganglia.

Much the same pupillary phenomena are seen during the action of drugs which affect the cerebro-spinal system, even in those which, in the first instance, produce myosis. In such, if the action of the drug be pushed, a stage is arrived at, when the function of the respiratory and cardiac centres is so seriously compromised that the pupils become widely dilated and fixed. This may be illustrated by the action of three drugs, opium, chloroform, and alcohol. When the physiological effect of a large dose of opium is fully established, the pupils are in a state of stabile myosis, and continue so as long as the cardiac and respiratory centres are active. When the dose is sufficiently large this stabile myosis is succeeded by a rapid change to that of stabile mydriasis, indicating that the function of the cerebrum and the medulla has been so dangerously interfered with, that it falls practically into abeyance, the cardiac action continuing for a brief period, partly through the inherent ganglionic cardiac centres, the respiration meanwhile being extremely slow, irregular, and gasping. If energetic measures are not promptly adopted, the life of the patient is measured by minutes. This has been verified by frequent instances. Though a most dangerous symptom, it is not absolutely hopeless, as is illustrated by a case of this kind which fell under the writer's observation, in which the patient was restored by means of artificial respiration continued for two consecutive hours (the stomach having previously been duly washed out). At the termination of an hour and a half, when a pause was made in the artificial respiration, in order to test the condition of that function, there was only one gasp, as if the air had just distended the lungs after their artificial compression. The pulse, at the same time, was a mere thread, difficult to perceive. At the end of another quarter of an hour, feeble, shallow respiratory efforts were made by the patient. The artificial breathing was carried on for a further period of fifteen minutes, during which the lividity of the face and surface of the skin cleared away, and after that the patient was able to breathe without further aid. Patient ultimately recovered, and was alive four years afterward. It was interesting to note that after she had recovered partial sensibility, and when she was able to breathe of her own accord, the pupils returned to the condition of myosis from that of mydriasis, in which they had been during the time that artificial respiration had to be continued; or, in other words, they remained dilated until the function of the brain had partially recovered.

This condition of stabile mydriasis setting in so rapidly, and taking the place of the myosis, shows that the opium has no longer the power of asserting its well-known influence on the pupil in the presence of the suspension of the brain function. The change indicates that death is imminent. The mechanism producing contraction of the pupil in these instances may probably be the congestion of the brain and its membranes, as well as the ocular vessels; and the rapid dilatation of the pupil to the anæmia coincident with the suspension of function.

It is probable that statements made in some text-books, regarding the action of opium, as occasionally causing dilatation of the pupils may be explained in this way. When death ensues after opium-poisoning, the pupils first relax somewhat, and then become medium. From this it will be seen, it is quite erroneous to suppose that the pupils will necessarily be found contracted in the cadaver after death from opium-poisoning. In six instances which have come under personal observation, the pupils were found to measure from four to six millimetres after death, although they measured about two millimetres shortly before death. It is right to remember that the apparent congestion of the internal organs in opium-poisoning may, in part, be due to the great fluidity and dark color of the blood.

The coma arising from alcohol, as was pointed out by me in 1879, may be distinguished from that due to other causes by the fact that when the patient is left lying undisturbed for twenty minutes, the pupils are in a state of myosis, pin-head contraction. If an attempt be then made to arouse the patient by shaking him, or pulling his hair, though he still remain quite comatose, his pupils will slowly dilate, until, if the physical irritation be continued, they become fully dilated. If the patient be then left undisturbed, the pupils remain dilated for a period of variable duration—minutes, as a rule, after which they begin to contract, doing so at a very slow rate until they reach their former dimensions. The time taken for the transition from the fully dilated stage to that of pin-head contraction, is from five to ten minutes, varying according to the intensity of the coma. In some rare cases, where the coma is very intense, the change is completed in less than five minutes. In instances in which the patient is not deeply comatose, the dilatation continues for ten minutes, and then contraction sets in at a very sluggish rate. In persons in whom the alcoholic coma is passing off, and who are merely insensible, and who can be temporarily roused, the dilatation of the pupil once established continues, or it may become smaller, though it does not again attain the same degree of contraction it formerly did. These observations have been subsequently verified in many hundreds of cases. In some uncommon instances of cerebral compression, through apoplexy or other cause, the contracted pupil will be slightly enlarged on stimulation (not full dilatation, as in alcohol), and very soon, in most, immediately thereafter, it will contract to its former size.

But when the dose of alcohol is poisonous, and the patient is about to succumb from its effects, the pupils, after being contracted as above, pass rapidly into complete stabile mydriasis. This is a condition which must be very seldom seen as arising from alcohol, but was once observed by the writer, in the case of a man who had half an hour previously drank off at a draught a quart of strong alcohol (proof strength); the pupils becoming widely dilated and fixed, the patient dying fifty minutes afterward. Here the function of the medulla had become interrupted, and finally suspended, just as in opium-poisoning.

In chloroform narcosis, the same pupillary phenomena are observed during the extreme stages. From personal observations, extending over several hundred carefully recorded cases, it is seen that the pupils are in a very variable state during the preliminary periods, much too variable to permit any rule being formulated regarding them. When, however, reflex action is abolished, except in the cardiac and respiratory centres, the pupils become contracted and fixed. This may be regarded as the safety zone of complete chloroform narcosis. But if the anæsthesia be pushed beyond this stage wide dilatation of the pupils ensues, indicating a suspension of function in the cardiac and respiratory centres. This is a most critical condition, though one by no means necessarily fatal, as by lowering the head and raising the floor of the table, and by promptly carrying out artificial respiration, even while feeble respiratory efforts are being made by the patient, the danger may be, and very frequently has been averted. Given a person free from organic lesion of the nerve centres, heart, or lungs, in whom during chloroform narcosis, stabile mydriasis suddenly occurs as a result of the action of chloroform on the respiratory centres, the patient ought to recover, if artificial respiration coupled with the lowering of the head, be promptly resorted to, and the former efficiently carried out. It is interesting to note in such cases the marked effect produced by elevating the foot of the table, so as suddenly to place the patient's head and thorax at a very low level, the pupils becoming quickly contracted. In this connection, however, it ought to be borne in mind, that one of the earliest indications of a return of reflex action is vomiting, which, as a rule, is accompanied by dilated pupils, the result of cerebral anæmia.

In cases where the functions of the cerebrospinal system, especially of the cardiac and respiratory centres, have already been enfeebled (by organic lesion or otherwise), chloroform acts more powerfully; a few whiffs sufficing to induce complete insensibility, and the administration of an ordinary dose giving rise to an alarming state, which only prompt measures can prevent from becoming fatal. In such cases the pupils very readily pass into wide dilatation with a very small amount of chloroform.

When the function of the brain is suspended by want of oxygenated blood, the pupils are widely dilated and fixed. This may be seen in cases of asphyxia either by poisonous gases or arising from mechanical causes. It was once observed by the writer in two men who had all but succumbed by inhaling coal-gas, emanating from a broken gaspipe in their bedroom; and it is also seen in cases of hanging.

Turning to the effects of pressure exercised upon the brain as a whole, it produces pupillary states varying according to its amount. When it is sufficient to place in abeyance the cerebral function, mydriasis occurs; and when of much less degree, causing only a small area to be involved, it is usually followed by myosis. The following is a typical case, illustrating these points as well as the most carefully planned and skilfully executed experiment could do.

While acting as House Surgeon in the Royal Infirmary, Glasgow, in 1870, the writer had the opportunity of observing the following case:

A well-developed and strongly built man, about twenty-eight years of age, was brought into the Infirmary in an unconscious state, having fallen from a height about an hour previously. There were bruises on his face, nose, and forehead, and there was slight oozing of blood from both nostrils. He was deeply comatose, his breathing was stertorous, his pulse slow and feeble, and the pupils of both eyes were widely dilated and fixed. Urine and feces had been passed involuntarily. This state continued for fully half an hour after admission, during which his pulse became so feeble, as sometimes to be scarcely perceptible; his breathing was even more deeply stertorous, and it was accompanied by lividity of the face. At the expiration of this period, a sudden gush of blood from both nostrils poured forth as a torrent, causing wide distention of the nasal alæ. This continued for full two minutes, then quickly lessened in quantity, becoming a mere trickling, which lasted half an hour. The pupils, which had been widely dilated and fixed from the time of his admission into the hospital up to the very moment when the blood began to escape in such volumes from both nostrils, were found at the expiration of two minutes, during which it flowed, to have become medium, to be quite active, readily contracting when exposed to light, and quickly dilating when shaded. Along with this, the lividity of the face disappeared, the breathing became free, though the pulse was extremely weak, and the patient pale. He however soon recovered consciousness, answered a number of questions correctly, and expressed surprise at finding himself in the infirmary. He also remembered, and was able to describe some of the circumstances connected with the accident which caused his injuries. This stage lasted two hours, the pulse and color of the face steadily improved, and during the whole time the pupils were active and normal. Half an hour later, the pupils began to be sluggish and contracted. Three hours after the hemorrhage they were very sluggish and contracted. Four hours later, they did not respond to light, though they were not so contracted as they had been half an hour previously. Patient no longer answered questions, but still was sensible to external impressions, especially of a painful kind. Six hours after the hemorrhage the patient was deeply comatose, and otherwise much in the same condition as when admitted, his pupils having again become widely dilated and fixed. Ten hours subsequently to the hemorrhage (ten and a half hours from admission) breathing became very labored, the lividity of the face marked, pulse flickering, and death ensued about twelve hours and twenty minutes after the hemorrhage from the nose.

The post-mortem examination showed that the cribriform plate of the ethmoid was extensively fractured, the aperture between the fractured bones being filled with clot; the crista gallæ was driven upward, the anterior part of the longitudinal sinus was ruptured, and a clot of blood existed between the dura mater and the skull, filling the greater part of the anterior fossæ, and pushing backward the brain. Both frontal lobes were much compressed, the convolutions were flattened, and the brain tissue comprising them was somewhat softened. The brain, as a whole, bore evidence of compression, but it was otherwise healthy. With the exception of a fracture of both bones of the leg, there were no other injuries.

REMARKS.—This case may be divided into four periods, each being accompanied by a peculiar pupillary condition. First, there was a period of severe intracranial pressure, indicated by symptoms of cerebral compression, in which the brain function was for the most part suspended. The pupils at this stage were widely dilated and fixed. This condition was present at the time of patient's admission, and half an hour afterward ended abruptly on the escape of blood from the interior of the skull. Second, with the relief of the intracranial pressure, the indications of compression of the brain disappeared, its functions returned, the patient regaining consciousness. The pupils then became normal, responding actively to the alternate action of light and shade. This state was the immediate sequence of the relief of the pressure on the brain by the escape of the pent-up blood, and it continued for two hours, until after the period of reaction had set in. Third, there was a period of slight cerebral pressure, due to the recurrence of hemorrhage within the skull, occurring shortly after reaction had set in. This was the direct result of the reaccumulation of blood within the skull, and the consequent pressure on the brain, a clot having filled the aperture in the fractured ethmoid. The patient in this stage had become drowsy. The pupils gradually lost their activity, becoming very sluggish and somewhat later contracted. This period lasted about four or five hours. It corresponded to slight cerebral pressure. Fourth, a stage of severe intracranial pressure ensued, marked by all the symptoms of cerebral compression and accompanied by widely dilated and fixed pupils. This lasted about four and a half hours, ending in death. This corresponded to the first stage on admission, the function of the brain having again become suspended. In a tabulated form it would appear thus:

Pressure.	*Action on Brain.*	*Effect on Pupil.*
1. Very severe.	Function almost in abeyance.	Stabile mydriasis.
2. Removal of pressure.	Restoration of function.	Normal and active.
3. Slight.	Interruption of function.	Contracted and sluggish.
4. Very severe.	Suspension of function, ending in its abolition.	Stabile mydriasis.

The same pupillary phenomena arising from pressure, induced by a pathological cause, are illustrated by a case of tubercular meningitis with its accompanying effusion, which is given elsewhere in detail. On admission, the patient, who was insensible, gave vent to the peculiar "hydrocephalic cry." She could move her limbs freely, her pupils were in a state of myosis (stabile). At this stage, it was probable that she suffered from interference with cerebral function, caused by effusion of fluid exercising a degree of pressure on the brain. A few days subsequently, she became unconscious, pulse very feeble, respiration labored and shallow accompanied by great lividity of the face, and a markedly subnormal temperature. The pupils were then in a state of stabile mydriasis. It was diagnosticated that the fluid had further increased, and was now exercising very severe pressure on the brain, placing its function in abeyance, with the exception, perhaps, of the respiratory and cardiac centres, which, though seriously interfered with, were still able to act to a slight extent. To relieve this pressure, a puncture was made through the brain membranes, when a large quantity of cerebrospinal fluid escaped in a jet. While the fluid flowed away, the respirations improved, the cardiac impulse increased in strength, the lividity disappeared, the pupils became much smaller, and shortly afterward became normal in size and sensitive to light, though they were somewhat sluggish. This condition continued for twenty-four hours, the temperature increasing from subnormal to 102.8° F. Ultimately the patient died.

The condition may be thus formulated:

Pressure.	*Action on Brain.*	*Effect on Pupil.*
1. Slight.	Interference with function.	Myosis.
2. Severe.	Function in abeyance.	Stabile mydriasis.
3. Removal of pressure.	Function nearly quite restored.	Mobile and almost normal.

Twelve cases of obliteration of function from pressure and bruising of the brain. Numerous instances of severe injury to the cranial contents have been observed by the writer in which both pupils were widely dilated and fixed; post-mortem examination afterward showing extensive destruction of the cerebral substance. In twelve such cases the patients died within a few hours after the accident. The post-mortem in these showed, besides serious injury of the skull, that both cerebral hemispheres had been encroached upon by extensive extravasations of blood, or by portions of bone or foreign matter driven into the cerebral substance, exercising severe and general pressure and producing extensive laceration of the brain. In all of them there was destruction of both hemispheres, sufficient to obliterate function.

Four cases of extensive bruising and laceration of the brain without pressure. Besides these, there were four cases in which a sufficient hiatus had been formed in the osseous walls of the skull to prevent the possibility of pressure being exercised on the hemispheres, and yet the pupils were dilated; the extensive laceration and severe bruising of the brain having destroyed the function of the part. Nor is the widely dilated and fixed pupils arising from cerebral pressure limited to surface lesions, or such as are caused by traumatism. In cases of extensive hemorrhage from the basal or deep cerebral arteries, causing great pressure or laceration of the brain, widely dilated and fixed pupils are seen, and they are accompanied by abolition of cerebral function. In one instance of this kind which came under personal observation, the lateral ventricles had become greatly distended with blood, the coma was profound, and both pupils were in a state of stabile mydriasis, this being the usual condition of the pupils in such lesions. In another instance, a large cerebral abscess had burst into the ventricles and this was notified by the sudden accession of deep coma, accompanied by widely dilated and fixed pupils. These phenomena are in accordance with those observed by others, who have pointed out that congestion of the membranes and slight hemorrhage into the ventricles is accompanied by contraction of the pupils, while profuse hemorrhage into the ventricles produces dilatation of the pupils. In the latter case, it is supposed that the result ensues from compression of the common motors, but the pressure no doubt causes suspension of function of the brain as a whole.[1]

It is true that cerebellar hemorrhage is generally indicated by contracted pupils, but this is not always so. When the pupils are contracted the extravasation is slight, but when the outpouring of blood is much greater, then dilatation ensues. It is not often that dilatation is seen from hemorrhage into this seat, on account of the great importance of the structures in the immediate vicinity, a comparatively small lesion producing a fatal result.

Both pupils dilated and fixed from very large unilateral lesions. In other instances both pupils were dilated and fixed, the lesion being unilateral, though indirectly the opposite side of the brain was implicated. Six cases of this kind were observed. All of them had sustained fracture through the middle fossa with rupture of the middle meningeal artery or one of its branches, from which extensive unilateral extravasation of blood had taken place. In a general way, these fractures may be described as extending from the base to above the level of the parietal protuberance, and the extravasated blood from the base of the anterior fossa to the posterior part of the middle fossa. The pressure was greatest opposite the middle lobe, the prominences of the convolutions being there effaced, and the outer wall of the lateral ventricles being flattened. In at least three of these the opposite hemisphere was involved directly by the pressure which was communicated through the falx cerebri, a slight concavity on the inner side of the opposite cerebrum indicating the seat of pressure. The opposite hemisphere, from the one flattened by direct contact with the blood clot, would likewise have been affected by the increased amount of cerebro-spinal fluid displaced by the blood clot from the other hemisphere, though the pressure from this cause could not be great, distributed as it would be over the whole cerebro-spinal axis. In these instances the patients survived the injury by only a few hours, during which they were plunged in profound coma, and therefore it is possible that the dilatation of the pupil on the side opposite the lesion might have resulted from suppression of brain function (concussion) on that side; though it is probable that the pressure communicated from the lesion in itself exercised a powerful action. It is further possible that the ablation of function by pressure of the white fibres of the centre of the hemisphere would in itself have produced the paralytic mydriasis of the opposite side, though this is much more prone to occur when the blood is extravasated into the interior of the brain.

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[1] M. Jordan states that "dilatation and total immobility of the pupils on the approach of a lighted candle is one proof of great engorgement of the brain in apoplexy." Dict. des Sc. Méd., 1878.

One pupil dilated and fixed, the other contracted, accompanied by lesions on both hemispheres. A few instances occurred in which, with lesions on both sides of the brain, one pupil was in a state of extreme dilatation with fixity, the other was contracted and fixed. In the first instance of this kind, there was a clot weighing not quite half an ounce on the external surface of the dura mater, over the right hemisphere, opposite the anterior ascending convolution, while there was extensive laceration of the middle lobe of the left hemisphere, with its accompanying extravasation of blood. The patient was insensible, and lived for eighteen hours. The left pupil was widely dilated and fixed, the right was contracted and fixed. The function of the left hemisphere had been abolished, while that of the right had only been partially interfered with. In the second instance there was slight laceration of the cortical substance, principally on the base of the first and second left frontal convolutions, with its accompanying extravasation; while on the right side there was an extensive blood clot which filled the subdural space, the dura mater having been ruptured by a spiculum of bone which had penetrated a branch of the middle meningeal. The patient was insensible, surviving the accident by only four hours. During life the right pupil was dilated, and the left was contracted, both being fixed. In the third case there was an extra-dural clot of blood weighing three ounces, situated principally over the outer portion of the left middle fossa; while on the right side, at the junction of the parietal with the occipital bone, near the vertex, there was a small depressed fracture, an osseous spiculum having penetrated the dura mater. This patient was insensible, and lived about forty-eight hours afterward. He had other bodily injuries of a severe kind, which, however, did not exercise any material effect on the brain-function. The right pupil was slightly contracted and very sluggish, while the left was dilated and fixed. In the fourth, there was a very thin diffuse layer of blood spread over the surface of the pia mater covering the convolutions in the anterior portion of the middle lobe of the left hemisphere; while on the right hemisphere there was an extensive laceration and extravasation into its middle and posterior lobes.

Patient lived a few hours after the accident, during which he was insensible. The right pupil was dilated and fixed, while the left was slightly contracted and sluggish. Some would ascribe these results to irritation and paralysis respectively of the oculomotor; while others would look upon them as due to vascular changes in connection with the cerebral lesions.

One pupil dilated and fixed, the other normal. Many instances have been observed of dilatation and fixity of one pupil, while its fellow, perhaps with the exception of a little sluggishness of movement, remained normal. These were all cases of fracture of the middle fossa of the skull, and in comparatively few of them was the diagnosis verified by post-mortem examination, though little doubt could exist as to the lesion when accompanied, as they were, by the usual phenomena of hemorrhage from the ear, followed by discharge of cerebro-spinal fluid, ptosis, occasionally external strabismus, facial hemiplegia, and paralysis of members. In such the patient was generally insensible at the outset when both pupils were dilated and fixed. As the patient recovered consciousness, one pupil became normal while the other remained dilated and fixed, this being on the side of the lesion. In some of these the visual power on the same side as the dilated pupil was in abeyence; in others, it was imperfect, and in a few it was almost normal. The pressure exercised by the blood-clot must have been sufficiently great to place in abeyance the function of the greater part of the affected hemisphere, as evidenced by the complete paralysis of the opposite member, the interference with vision, and the evident effect upon the third nerve. In those cases in which post-mortem examinations were obtained, the basal fracture was continued into or toward the vertex, and the clot in most instances occupied the whole of the middle fossa from the petrous portion of the temporal to the vertex, the convolutions, especially the ascending, being considerably compressed. The opposite hemisphere was in almost a normal condition. In those that recovered, the dilatation gave way slowly, and was followed by a degree of contraction along with sluggishness of movement which ultimately passed off; though in one case it still remained small and sluggish at the termination of the fifth month from the date of the accident. These changes were evidently determined by the gradual absorption of the blood-clot. It is more than likely that in most of them the third as well as the seventh nerve was paralyzed, and in one or two instances the fifth in addition, as indicated by the loss of sensation on the affected side. Occasionally the second was interfered with. Hearing was generally much affected, if not absolutely in abeyance on the affected side, during the early months, mechanical obstruction by blood-clot producing loss of hearing as well as interference with the nerve.

Pupil contracted on only one side. Quite a number of cases have been observed by the writer in which, while one pupil was normal, its fellow was contracted and fixed, and where, judging from other symptoms, a cerebral cortical lesion was diagnosticated as existing on the same side as the affected pupil. In at least three of these instances the diagnosis was verified by demonstration of the lesion on the operating table, and by the fact that its removal was followed either by great amelioration of the state of contraction, or by its complete relief. One was a case of fibrous tumor of the dura mater, situated over the left anterior lobe, which exercised pressure on the surface of the first and second frontal convolutions. The pupil on the same side as the lesion was minutely contracted and fixed. Operation was demanded for epileptoid seizures. The tumor was removed, and the patient recovered. The activity of the pupil very soon returned, and the contraction was in great measure removed within a few days subsequent to the operation. Ultimately the two pupils acted alike. The second was a case in which there was a cortical extravasation of blood from a bruise of the ascending frontal convolution, and which was surrounded by an area of irritation. The pupil on the same side was contracted and fixed. The lesion was removed by operation. The patient recovered, and the contraction, though at first remaining to a slight extent, disappeared completely within a month. In the third instance, the dura mater over the right frontal region was punctured by an osseous spiculum. The pupil of the corresponding side was minutely contracted. On the removal of the fragment of bone, and the escape of a small quantity of fluid blood from under the dura mater, the contraction of the pupillary sphincter relaxed immediately. Four hours afterward the pupil responded slowly to alternate light and shade. Two days after operation it was quite normal. There are many explanations advanced for such pupillary effects arising from these lesions. The influence of the dura mater on the basal nerves has to be borne in mind, especially when it is inflamed; also that of the possible effect which might be transmitted through the fibres of origin of the optic to its centre, and so to the third nerve; and, again, the effects of irritation arising from pressure exercised on the two alleged centres of pupillary movement in the cerebral cortex. All of them could, however, be more directly explained by vascular changes accompanying these lesions producing their effects upon the vessels of the eyeball.

It is interesting to note that in cerebral softening the action is like that of hemorrhage into the brain, producing abolition of function and dilatation of the pupil. In some such cases there is no doubt that the oculomotor nerve has also lost its power, as indicated by the loss of ocular movement; but in others the movements are retained, the pupil, however, being dilated.

The effects of spinal lesions on the pupil. There have been very many instances under personal observation of spinal irritation inducing labile mydriasis, and there have been a few of complete lesion of the cord causing stabile myosis. Of the latter, one occurred in a man of fifty years, the subject of a sarcoma involving the bodies of the lower cervical vertebræ pressing upon the spinal cord and ultimately crushing it. The pupils were about two millimetres in diameter, and fixed. The vessels of the head and face were dilated, and the conjunctivæ were suffused. One was a child, aged five years, affected with tubercular infiltration and destruction of the bodies of the third, fourth, and fifth cervical vertebræ, opening up the canal and exposing its contents. An abscess formed, which involved a limited area of the spinal canal opposite the affected vertebræ. The anterior portion of the theca was represented by a fungated process of granulation tissue; the cord was in a much softened condition, and was surrounded with pus. The pupils, which for months had been in a state of labile mydriasis, became, during some days prior to death, contracted to two and a half millimetres in diameter. Several traumatic lesions of the cord in the upper dorsal and lower cervical vertebræ have been observed, accompanied by myosis, the contraction being from two to three millimetres. In each of those death ensued within some hours or days from date of injury. In all there was destruction of the cord at the seat of injury; in some, to the extent of severance of almost its entire calibre.

In the foregoing there is sufficient evidence to show that the suspension or abolition of cerebral function in the living body is attended by mydriasis, the latter being the sequent of the former. If inquiry be made concerning the mechanism inducing this pupillary effect coincident with the arrest of cerebral function, the theory which explains the greater part, if not the whole of the phenomena, is that which has been so ably advocated by Mosso. The passive movements of the pupil are regulated by the vascular system of the iris, which is in complete harmony with that of the encephalon. In those conditions inducing general suspension of the cerebral function, a state of ischemia prevails in the brain and iris inducing mydriasis. This, likewise, obtains in unilateral lesions, where the pressure is so great as to induce anæmia of brain and iris. Myosis may also be brought about by a like mechanism acting in the opposite direction. The "irritation" setting up congestion of the cerebral and meningeal vessels, leads to congestion of the vessels of the iris, and so produces contraction of the pupil.

When investigating the cause of a given pupillary state, this should be done in a methodical manner, examining the various sources controlling pupillary movements, and eliminating those which do not apply. The examination ought to be conducted by answering, *seriatim*, the following questions:

1st. Is the pupillary condition due to the local or constitutional action of any drug?

2d. Is the state of the pupil dependent upon any local ocular lesion or optical defect (including artificial eyes)?

3d. Is it due to any spinal or sympathetic lesion, more especially of the cilio-spinal region and the cervical sympathetic?

4th. Is it dependent upon any localized cerebral lesion affecting special brain centres—*i. e.*, corpora quadrigemina, optic thalamus, or the origin and intracranial course of the second, third, and fifth nerves, especially the third?

5th. If due to none of the above, the probability is that it depends upon either a suspension of brain function or to some cerebral "irritation," in either case inducing vascular changes in the encephalon and iris.

The following points, among others, may be formulated from the foregoing:

(A) 1. When the function of the brain is in abeyance, the pupils are in a state of stabile mydriasis.

2. This may arise either from temporary suspension or from abolition of function.

3. Temporary suspension is illustrated by shock, and the effect of some poisons; while the abolition of function is exemplified by extensive laceration and compression of the brain.

(B) 4. When the function of the brain is interfered with by conditions usually included under the term "irritation," the pupils are in a state of myosis; sometimes labile, but generally stabile myosis.

5. This "irritation" or interruption of function may be seen during certain degrees of cerebral anæmia, produced experimentally, and not as a pathological result; certain amounts of brain pressure, and certain stages of intracranial inflammation.

6. These are illustrated in persons who have suddenly lost a considerable quantity of blood (about a fifth of the whole); in the growth of intracranial tumors and the formation of sanguinolent serous and purulent effusions, when the degree of pressure may be denominated as "medium," and at certain periods of meningitis and encephalitis.

(C) 7. The same pathological factors which cause myosis may also cause mydriasis, the degree in which these factors are present being the determining point between the former and the latter, and not *merely* the particular locus in the brain.

8. It is well illustrated by cases where the hemorrhage is repeated, and is finally pushed to syncope; in intracranial pressure, which is gradually increased until it becomes great, such as arises from tumors, blood clots, and inflammatory products.

(D) 9. When the function of one-half of the cerebrum is placed in abeyance by a superficial or cortical lesion, the pupil on the same side as the lesion is in a state of stabile mydriasis.

10. This is well illustrated in cases of intracranial sanguinolent effusion consequent on injury (see list of cases).

(E) 11. When the function of one-half of the cerebrum is interfered with by some source of cortical irritation, the pupil on the corresponding side to the lesion is in a state of myosis.

12. This is illustrated by traumatic and pathological lesions affecting the cortex of the cerebrum (see list of cases).

(F) 13. Hemorrhage into the pons Varolii when small, causes strongly contracted pupils; but when it is more extensive, involving the gray matter beneath the aqueduct of Sylvius, a state of stabile mydriasis is induced.

14. Effusions into the lateral ventricles when small, produce contraction of the pupils, but when the effusion is great stabile mydriasis ensues.

15. Inequality of the pupils indicates a unilateral lesion or lesions.

16. When the lesion is cortical and unilateral the pupillary manifestations are on the corresponding side. When the basal nerves are affected unilaterally the pupillary effect is manifested on the same side as the lesion. When the lesion is unilateral and affects the function of the white fibres of the cerebrum the opposite pupil is generally affected. When the basal ganglia are implicated unilaterally the pupil is sometimes affected on the same side as the lesion, occasionally on the other side.

In a case of cholesteatoma and in another of glioma of the right optic thalamus, dilatation of the left pupil was found (*Ross*, vol. ii. p. 572).

In lesions of the cerebral peduncles the pupil is affected on the same side as the lesion.

Lesions in the corpora quadrigemina affect both pupils, irritation[1] causing contraction, destruction causing dilatation and immobility.

Section or destruction of one optic tract, causes dilatation of the opposite pupil and blindness of the opposite eye.

17. Irritation of the cord, especially the cilio-spinal axis, produces dilatation of the pupils, while destruction of the cord causes contraction. These effects are generally seen in both pupils, though, experimentally at least, they may be confined to the same side as the lesion.

18. The pupils are affected in the same way by lesions of the sympathetic, though in unilateral lesions it is only the pupil on the same side as the lesion which is affected.

19. Speaking generally, when myosis is due to a cerebral cause, it indicates the earlier stages of various affections; when due to a spinal

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[1] Unilateral electrical stimulation causes dilatation of both pupils, the opposite one becoming first dilated. Ferrier explains this as the result of irritation of a sensory structure, in this case acting through the medium of the anterior roots of the second dorsal nerves which ascend in the cervical sympathetic.

lesion, it points to a most serious paralysis, often to the destruction of the part. When mydriasis arises from a cerebral lesion it is generally present in large amount; when due to a spinal affection it indicates irritation of the part.

MYOSIS OCCURS UNDER THE FOLLOWING CONDITIONS:

1. When a bright light acts upon the retina.
2. Accommodation for a near object.
3. Rotation of the eyeball inward.
4. Local irritation or painful affections of the eyeball.
5. Irritation of the oculomotor nerve.
6. Paralysis of sympathetic roots of lenticular ganglion or trunk of sympathetic in the neck. In paralysis of the fifth there is myosis and inflammation passing on to destruction of the eyeball.
7. Paralysis of the ciliospinal region of spinal cord. All affections which destroy the cervical spinal marrow and intercept its conductibility produce congestion of the face and contraction of the pupils. In neurosis, which suspends or diminishes the tone of the sympathetic or spinal axis.
8. Encephalic congestion, such as: obstacle to return of blood in jugulars; venous congestion due to cardiac causes; active hyperæmia, plethora, fevers, pneumonia, hepatitis, etc.; when animal is suspended by the heels; in early stages of meningitis and encephalitis; in acute mania with marked activity of the cerebral circulation; in chronic mania pupils are variable, when contracted are said to indicate supervention of paralytic dementia.
9. During sleep; some believe this to be due to the congestion of the cerebral vessels and those of the iris (Mosso); others, to the inward rotation of the eyeball.
10. In the early stages of cerebral tumor.
11. In small hemorrhages into the cerebellum. In irritation of the cerebellum, contraction of the pupil on same side as lesion ensues.
12. Electrical stimulation of the angular gyrus frequently causes contraction of the pupil.
13. During forced expiration, when the eye is at the same time passive. Also generally seen during the period of apnœa in Cheyne-Stokes respiration.
14. Convulsions arising from meningo-encephalitis are said to be accompanied by myosis, while in convulsions due to epilepsy and in epileptiform fits they are usually accompanied by mydriasis.
15. When the eye contracts on accommodation to a near object, yet does not contract to light, this indicates a lesion situated between the corpora quadrigemina and the oculomotorius. This affection is known as the Argyle-Robertson symptom. It is seen in locomotor ataxia and occurs in the progressive paralysis of the insane.
16. During uræmic coma.

17. Myotics: physostigmine, nicotine, pilocarpine, morphine, muscarine.

MYDRIASIS OCCURS UNDER THE FOLLOWING CONDITIONS:

1. In darkness or in subdued light.

2. Accommodation for distant objects.

3. Rotation of the eyeball outward.

4. In forced movements discharged from the medulla: vomiting, swallowing, chewing, forced respiration.

5. Paralysis of the oculomotor (accompanied or not by immobility of eyeball, external strabismus, diplopia, etc.).

6. Destruction of the optic: amaurosis. When unilateral, associated movements continue.

7. Irritation of sympathetic: powerful impressions on sensory nerves; strong moral emotions, mental pain, grief, fear; neuralgia of the fifth nerve.

8. Irritation of the spinal cord, especially ciliospinal region.

9. Encephalic anæmia: In all cases where there is reflex contraction of the vessels of the head; when loss of blood from the body is excessive; obstruction of the carotid arteries; in thrombosis of brain sinuses; dilatation of mesenteric vessels when extreme; syncope, intense cold, rigors; dyscrasias of the blood, convalescence, cachectic conditions; asphyxia, epilepsy, in certain stages of these affections.

10. Pressure of cerebrum when great in amount, as from hemorrhage, neoplasms, etc. In the last stages of meningo-encephalitis.

11. In cerebral softening. In acute dementia (œdema of cortex cerebri) observers state that the pupils are invariably dilated (Hutchinson).

12. In idiots the pupils are generally dilated.

13. During deep inspiration, generally in respiratory period of Cheyne-Stokes breathing.

14. Hemorrhage into centrum ovale and into cerebral peduncles.

15. Ferrier produced dilatation of opposite pupil by destructive lesion of the optic tract in the thalamus, indicative of rupture of the centripetal fibres to the irido-motor nucleus in the floor of the Sylvian aqueduct.

16. In hydrophobia there is mydriasis.

17. Mydriatics: atropine, homatropine, duboisine, daturine, hyoscyamine. Curare injected subcutaneously in animals (five to ten centigrammes) induces in one or two hours complete paralysis of the third nerve.

THE EFFECT ON THE PUPIL OF LOCAL CONDITIONS OF THE EYEBALL:[1]

1. Hyperæmia of the iris produces contraction of the pupil which darkness scarcely diminishes.

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[1] The writer is indebted to Dr. Thomas Reid for revising this list of pupillary effects occasioned by local conditions of the eyeball.

2. Presbyopia and hypermetropia cause contraction of the pupils in cases where continuous and excessive strain for near accommodation has been long continued and has produced asthenopia.
3. Pupillary atresia, consequent upon chronic irritation with posterior synechia, producing contraction of the pupil.
4. In synechia total dilatation is impossible, the iris only dilating where free, hence, the pupil is irregular. If the synechia is annular the pupil is both contracted and immobile.
5. In microria there is a congenital state of extreme contraction.
6. In glaucoma the pupil is dilated, contracting little or not at all to the action of calabar bean.
7. In coloboma, both in the congenital form and after iridectomy, there are irregularity and immobility of the pupil.
8. In idiopathic mydriasis there is little contraction to the action of light or to myotics.
9. In certain cases of amblyopia and amaurosis there is dilatation of the pupil.
10. In hippus pupillæ there are alternate contraction and dilatation often accompanied by nystagmus.
11. Inequality of the pupils exists in some who have different degrees of refraction in the two eyes, one being emmetropic and the other myopic.

THE THERMAL DEATH-POINT OF PATHOGENIC ORGANISMS.

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An exact knowledge of the thermal death-point of pathogenic organisms is desirable, both as a matter of general scientific interest, and from a practical point of view. As biologists, we wish to know whether the vital properties of the living protoplasm contained in the minute vegetable organisms in question are destroyed at a uniform temperature, and if so at what temperature; or whether there is a considerable range in the limits of vital resistance to heat exhibited by different organisms of this class. As sanitarians, we wish to know what temperature can be relied upon for the destruction of disease germs in the excreta of patients suffering from typhoid fever, from cholera, and from other infectious diseases transmitted by means of the alvine discharges of the sick; whether boiling of infected clothing, or of drinking water contaminated with disease germs, is a safe means of disinfection, etc.

Various experimenters have recorded observations with reference to the thermal death-point of different microörganisms, but, so far as I